cians provide care through their offices for obstetrical or inpatient hospital care on a rotating basis, charging Medi-Cal for their services. The clinic staff does the necessary paperwork and phone calls to obtain treatment authorization from Medi-Cal when hospital admission or surgery is required.

This approach has proved quite successful. Patients are assured the availability of care, and physicians are able to fulfill their desire to do their share of charity care without burdening their offices with Medi-Cal paperwork. The clinic itself has been able to generate enough charges, even at low Medi-Cal reimbursement rates, to pay the overhead and personnel costs.

There is no doubt that the current Medi-Cal program is inefficient, expensive and filled with impediments to reimbursement for provision of medical care. These problems can be solved in part by using physicians' traditional willingness to provide some free care as part of their professional obligation.

JACK SAUNDERS, MD
Medical Director
Mt Shasta Medi-Cal Clinic
908 Pine St
Mt Shasta, CA 96067

REFERENCE

1. Boni BM: Abolish Medi-Cal (Correspondence). West J Med 1985 Apr; 142:559

Dysthyroid Ophthalmopathy

TO THE EDITOR: We read with interest the article "The Ophthalmopathy of Graves' Disease" in the April 1985 issue.[1] Our experience with several hundred patients with dysthyroid ophthalmopathy has allowed us to formulate a protocol for their management.

During the acute phase, we recommend several supportive measures, including elevation of the head of the bed and artificial tear drops to the eyes. In addition to corneal exposure from proptosis and eyelid retraction, many of these patients have decreased tear production. We feel that control of any thyroid gland abnormalities is important to the general health of the patient, but have not seen any correlation between this control and the progression of the orbitopathy. In general, the orbitopathy tends to run its course. Medicines, surgery and even radiation may make the patient more comfortable, preserve the vision and more rapidly restore function. Nonetheless, the disease process itself must run its course until quiescence is achieved. Prednisone is occasionally of use in patients with severe inflammatory reactions, but should not be used on a regular basis and should not be used for longer than several weeks.

During the acute phase the patient should be examined regularly by an ophthalmologist for evidence of dysthyroid optic neuropathy. This is most likely caused by the enlarging extraocular muscles compressing the optic nerve at the apex. It is interesting to note that in patients with maximal posterior muscle enlargement there is minimal proptosis but often optic neuropathy develops. When optic neuropathy is detected, we institute a trial of corticosteroid therapy for several weeks. Oral administration of prednisone may minimize orbital apex edema and allow the patient to pass through this phase of the orbitopathy without surgical intervention or irradiation. If optic neuropathy persists in a patient on a regimen of 100 mg of prednisone a day for several weeks, surgical orbital decompression is carried out. Careful attention is given to decompression of the orbital apex. It is only after failure of corticosteroids and surgical decompression to relieve the optic neuropathy that we advise radiation therapy. In management of more than 150 cases we have used radiation therapy in three patients. In our experience, irradiation causes extreme fibrosis of the orbital contents and increased difficulty with the patient's future rehabilitation. We strongly feel that radiation therapy for nonmalignant maladies is to be avoided when possible.

Once the acute phase has passed and the patient is stable for six to nine months, surgical rehabilitation may be considered in a stepwise fashion. We feel that there are four sequential stages to this rehabilitation. Stage one is orbital decompression to move the eyes back to a more posterior and protected position in the orbit. Stage two is an extraocular muscle surgical procedure to correct diplopia. Stage three is eyelid malposition correction (such as recession of eyelid retraction). Stage four is removal of excess eyelid skin and fat that has formed as a result of the disease process. Each stage must be considered in order. Any unnecessary stage may be skipped. Surgical rehabilitation done in this order provides better results and keeps the number of procedures to a minimum. For example, strabismus operations carried out on patients with proptosis recess the muscles and allow the globes to become more proptotic. Subsequent orbital decompression moves the eyes and muscle cones posteriorly and often results in a change in muscle cone position with associated induced strabismus. The patient then requires an additional strabismus operation. Movement of the globes and muscle cones (decompression) results in a change in the position of the extraocular muscles and eyelid retractors. Movement of the extraocular muscles results in a change in position of the eyelid retractors. The logic of the four stage approach to surgical rehabilitation, therefore, is clear. We have been very pleased with the results when this staged approach is used.

NORMAN SHORR, MD
Division of Ophthalmic Plastic and Reconstructive Surgery
Department of Ophthalmology
Jules Stein Eye Institute
UCLA School of Medicine
Los Angeles, CA 90024

STUART R. SEIFF, MD
Division of Ophthalmic Plastic and Reconstructive Surgery
Department of Ophthalmology
UCSF School of Medicine
San Francisco, CA 94143

REFERENCE

1. Rapoport B: The ophthalmopathy of Graves' disease—Medical Staff Conference, University of California, San Francisco. West J Med 1985 Apr; 142:532-536

Familial Hiatal Hernia

TO THE EDITOR: In his letter in the November 1984 issue,[1] Dr Leung reports on a family in which six members were affected with a hiatal hernia. He goes on to observe that this may be the first recorded example of the familial occurrence of the disorder. While this may be true of the United States and Canada, it is certainly not true of the European literature.

In 1970 I published with P. Froggatt a paper[2] drawing attention to the etiologic role of heredity in hiatal hernia. In that article we undertook a comprehensive review of the world literature and reported on a family with eight unequivo-